MEDICAL PRACTICE

Clinicopathological Conference

A Patient's Life

DEMONSTRATED AT THE ROYAL COLLEGE OF PHYSICIANS OF LONDON

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The tenth of the quarterly series of clinicopathological conferences was held at the Royal College of Physicians of London on 30 January 1975 with Professor M. H. Lessof in the chair. The case was presented by Dr. C. S. Ogg and discussed by Dr. W. Cattell.

Clinical Summary

The patient was a housewife, born in 1941, who developed ankle oedema and proteinuria during her first pregnancy in 1963. In November 1965 she was admitted to Guy's Hospital for further investigation. At this stage she had moderate dependent oedema, blood pressure 160/120, microscopic haematuria, heavy proteinuria with hypoalbuminaemia (serum albumin 23 g/l), anaemia (haemoglobin 8·7 g/dl), and moderate renal functional impairment (plasma urea 11·0 mmol/l (66 mg/100 ml)), creatinine clearance 57 ml/minute).

She was treated with diuretics, steroids, azathioprine, and norethandrolone but she became more oedematous, her renal function fell (creatinine clearance 16 ml/minute) and her blood pressure rose further to 210/135. In January 1966 her azathioprine and norethandrolone were stopped but her steroids were continued for a further six months. Despite hypotensive and diuretic treatment (pempidine, methyldopa, and frusemide), her blood pressure was not well controlled. During the next 18 months she was admitted to hospital repeatedly because of uncontrolled hypertension with papilloedema, dependent oedema, anaemia requiring transfusion, and deteriorating renal function.

In July 1967 she complained of pains in her hands and developed swelling of the interphalangeal joints of the left thumb and of the distal interphalangeal joints of the left middle and right index fingers. At that time her plasma calcium was 1·88 mmol/l (7·5 mg/100 ml), phosphate 3·49 mmol/l (10·8 mg/100 ml), uric acid 0·36 mmol/l (6·0 mg/100 ml), Rose-Waaler test negative, antinuclear factor absent B1C globulin 200 mg/l. *X*-ray films of her hands showed periarticular calcification but no subperiosteal erosions. Her blood urea and plasma creatinine were 41·5 mmol/l (250 mg/100 ml) and 796 μmol/l (9·0 mg/100 ml) respectively.

She was started on haemodialysis, initially in hospital with a Kiil dialyser twice a week for 14 hours. Her hypertension was controlled moderately well (160-180/100-110) and hypotensive drugs were withdrawn. Nevertheless, she had recurrent problems with her shunt, requiring low dose treatment with warfarin, and because of continuing severe anaemia she was transfused repeatedly (450 ml every two weeks). She had had amenorrhoea since 1966, but in August 1968 she developed menorrhagia, which was exacerbated during dialysis. Her haemoglobin fell to 3·0 g/100 dl and she was admitted for blood transfusion and dilatation and curettage. Histology of the curettings showed a cystic proliferative endometrium and she was given a short course of Minovlar (norethisterone acetate 1·0 mg, ethinyl oestradiol 50 μg).

HOME DIALYSIS

In 1969 she was transferred to home dialysis, receiving three ten-hour treatments weekly. Her general condition improved and she was able to maintain a haemoglobin of 6-7 g/dl without regular transfusion. Her blood phosphate was maintained below 1·94 mmol/l (6·0 mg/100 ml) and the periarticular calcification diminished though arterial calcification persisted or worsened. *X*-ray films of her hands showed a few subperiosteal erosions, and her serum parathyroid hormone level was moderately raised (2·6 μg/l). Once again, however, she developed menorrhagia which was treated with norethisterone 10 mg daily. When this proved ineffective, hysterectomy was advised but refused, because of her hope that one day she might have a transplant that would allow her to have another child. During the rest of her life she was treated more or less continuously with Norinyl 2 (norethisterone 2·0 mg, mestranol 100 μg), two tablets daily.

During the next four years she remained reasonably well and was able to do a part-time job as well as her duties at home. However, from time to time she had vaginal bleeding requiring transfusion. Towards the end of 1973 her blood pressure started to rise once more and proved difficult to control by salt restriction. Bilateral nephrectomy was therefore carried out in January 1974 without incident. Nevertheless, during the months after the operation she was much more anaemic than before (haemoglobin 3·6 g/dl) and required repeated transfusion.

Her white count and platelet count were both slightly depressed at $4{\cdot}3 \times 10^9$/l and 158×10^9/l and red cell survival studies showed a greatly diminished half life (6·1 days) with significant sequestration in the spleen. A ^{99m}Tc colloid scan showed that the spleen was slightly enlarged and splenectomy was performed on 2 July 1974. The spleen was friable and after operation she developed a left subphrenic

haematoma which became infected and required drainage. While still in hospital she developed sudden severe abdominal pain, abdominal distension, and shock. Emergency laparotomy showed a haemoperitoneum due to rupture of the right lobe of the liver. The bleeding area was packed and the abdomen closed. Hepatic arteriography revealed multiple areas of abnormal circulation in both lobes of the liver. Bleeding continued and the liver was packed again on 3 August. The next day her abdomen was opened for the third time in an attempt to control the haemorrhage by ligating the hepatic artery. This was unsuccessful, however, and she died during the operation.

DR. CATTELL (1): This woman was 22 when she presented with a renal lesion during pregnancy. This persisted post partum and in 1965 she had the full-blown nephrotic syndrome. Quite properly she had a renal biopsy.

BIOPSY FINDINGS

DR. G. A. K. MISSEN (2): Light microscopy of thin paraffin sections showed every glomerulus to be abnormally solid with an excessively lobulated outline and proliferation of its mesangial cells. Visible capillary lumina were few and small. There were very occasional capsular adhesions, but no crescents. With silver-staining, mesangial matrix was seen to be greatly increased and to extend out from the centrilobular zones so as to enclose the capillaries and separate them from the overlying glomerular basement membrane, which appeared normal. At several points broad zones of hyaline eosinophilic material, retrospectively identifiable as large immune deposits, separated the basement membrane from the mesangial matrix. The name of this disease has changed more than once in the last 25 years; originally lobular and later membranoproliferative glomerulonephritis, we now call it mesangiocapillary glomerulonephritis.

DR. CATTELL: The biopsy findings are very relevant to the prognosis. In mesangiocapillary glomerulonephritis it is relatively bad with slow or rapid loss of kidney function. It has been described as hypocomplementaemic glomerulonephritis. The third factor of complement is commonly low in the acute phase of glomerulonephritis but, if this persists, it should raise the suspicion of mesangiocapillary glomerulonephritis.

What of treatment? I can't fault the diuretics. We now know that steroids and azathioprine singly or together are of no value in mesangiocapillary glomerulonephritis. In recent years Kincaid-Smith and her colleagues have introduced "the cocktail" —which is a combination of steroids; azathioprine or cyclophosphamide; heparin or warfarin; and a platelet stabilizing factor such as dipyridamole. In patients with rapid loss of kidney function who have an excess of epithelial crescents, this cocktail would seem to be of real value. Biopsy in this patient did not show significant crescent formation and on present evidence she would not be expected to benefit.

There is less evidence for the value of anabolic steroids such as norethandrolone. This may have relevance later on. The hypertension was not well controlled. This, of course, is disastrous, for uncontrolled hypertension accelerates loss of kidney function. It is rare to be unable to control the blood pressure with modern drugs, such as propranolol, diazoxide, or minoxidil.

She then had small joint involvement. May we see the *x*-ray films of that now? These show soft tissue calcification in relation to the finger joints. The joints themselves are normal with no erosions. Is this ordinary rheumatoid disease? It does not look like it and the patient is seronegative. We are told that the calcium/phosphate product is over 80. I suggest that this is a form of arthropathy found in association with calcification of soft tissues. This is also seen in sarcoid and in systemic sclerosis. Could it be pyrophosphate arthropathy which is not uncommon in renal disease? This affects large joints and produces calcification of the cartilage itself. Pyrophosphate crystals are found in the joint fluid. No, I think that this arthropathy is a consequence of soft tissue calcification.

Soft tissue calcification is not uncommon in chronic renal failure, particularly in patients on dialysis. Calcification in vessels may limit blood access for regular dialysis and hinder the transplant surgeon. So we try to prevent soft tissue calcification. The problem is usually the phosphate level, which in this case was unacceptably high. This should be controlled by giving Aludrox by mouth.

With haemodialysis there was moderate control of hypertension, to the extent that hypotensive drugs were withdrawn. It is not unusual to have problems with blood pressure control in the first five or six months of dialysis but these usually resolve and do not recur.

Shunt clotting and infection were common problems in the days of silastic arteriovenous shunts for access to the circulation. Because of shunt clotting, the patient was put on warfarin, though she was already anaemic and this could have increased her menorrhagia. Amenorrhoea is a common complication of renal failure in women. Effective dialysis usually reverses this, and indeed the return of menstruation is one of the best criteria for the adequacy of the dialysis. We also have faced the problem of menorrhagia. Patients on regular dialysis must have systemic heparinization two or three times a week and this may exacerbate vaginal bleeding if they have the potential for menorrhagia. Our patient did not want a hysterectomy because she hoped one day to have another child. Is this pie in the sky? The table shows that her expectation was not unrealistic. But how do you control the menorrhagia? Mr. Coltart will tell us that.

*Pregnancies in Women on Dialysis and with Transplanted Kidneys**

	Dialysis	Transplant
Abortion: Spontaneous	14 (50%)	10 (19·6%)
Surgical	11 (39·2%)	25 (49%)
Living child: Abnormal	1 (3·6%)	0
Normal	1 (3·6%)	13 (25·5%)
Still pregnant:	1 (3·6%)	3 (5·9%)
Total	28	51
Total women aged 15-49	9294	2185

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CONTROL OF MENORRHAGIA

MR. T. M. COLTART (3): The cystic proliferative endometrium suggests that she was not ovulating. Oestrogen stimulation will build this endometrium up and make any uterine bleeding more severe. It is a question of assessing risk factors. The only conservative possibility is to try an oral contraceptive, which these patients tolerate well, though there is a risk of hepatic dysfunction. 50 micrograms of oestrogen controls menorrhagia in most patients. Unfortunately to make it active by mouth it has to have an alkyl group on the 17-C atom and this is important in liver function. A progesterone-only pill may be tried but there is not complete cycle control, and break-through bleeding may occur. Moreover, ovulation is not always suppressed, which is unacceptable if contraception is required. The newer mini-pills, with only 30 micrograms of ethinyl oestradiol, would probably be tried first nowadays.

DR. CATTELL: Low-dose oestrogens also constitute our approach to the control of menorrhagia and are usually successful. On home dialysis her general condition improved. The phosphate was below 1·94 mmol/l (6·0 mg/100 ml) and her calcium/phosphate product stayed below 70. Professor Berlyne noted many years ago that soft tissue calcification occurs—he was describing the "red eye" lesion—if the product exceeds 70, but we suggest that even this is a high figure and attempt to keep it around 50. Her periarticular calcification diminished, though arterial calcification persisted or grew worse. By this time she had subperiosteal erosions, so she had developed secondary hyperparathyroidism. Did she have any subperiosteal erosions before starting dialysis?

DR. OGG (4): No. They were never a prominent radiological

feature and we had to look quite hard for them. There was just a short period in 1969-70 when there were obvious erosions.

BONE DISEASE

DR. CATTELL: Bone disease may be one of the major headaches in patients on regular dialysis. It has been said that all patients on regular dialysis treatment will develop radiological evidence of bone disease after four years. This is grossly overstating the case: incidence varies extensively throughout Britain, with areas of high incidence—for instance, in Newcastle upon Tyne and in the south west. We now think the bone disease is treatable—either medically, using calcium and one of the vitamin D products (recently 1-alpha-hydroxycholecalciferol and 1-25-dihydrocholecalciferol)—or occasionally by subtotal parathyroidectomy. My question about her bone status before dialysis was because we think that many people who get into trouble with bone disease already had it before starting regular dialysis.

She was reasonably well for the next four years, though she had to have many further transfusions. The policy throughout Britain is one of minimal transfusion because of the risk of iron overload and of serum-transmitted hepatitis.

The rise of blood pressure at the end of 1973 was one of the most difficult aspects of the case. It is extremely unusual to have late development of uncontrollable hypertension. I have never seen it and cannot find it reported in a quick review of the literature. The only possible explanation is that the patient was no longer restricting her sodium intake and had become much more casual in her use of salt. After some years on dialysis many patients can tolerate more salt, and this has been rather facilely attributed to autonephrectomy and the secretion of less renin. Could she have developed a phaeochromocytoma or Cushing's syndrome?

DR. OGG: No, I don't think so. The hypertension was rather more apparent than real, in that we were then demanding a lower blood pressure than before. But I am sure she was being casual with her salt intake, which was concealed by a simultaneous loss of flesh.

DR. CATTELL: The patient then had a bilateral nephrectomy. This is a most unfortunate step in the management of patients on regular dialysis.

ANAEMIA

Anaemia has been a consistent feature of this history. The principal defect which results in normochromic anaemia in chronic renal failure is impaired erythropoiesis due to a relative or absolute deficiency of "erythropoietin" production. That the kidney is important for erythropoiesis in the human is shown by many reports that the haemoglobin levels are lower and the transfusion requirements higher in anephric patients on regular dialysis than in those who have not had nephrectomies. Thus we are extremely loth to carry out nephrectomy in patients on regular dialysis, whether they are subsequently going to have transplantation or not.

What can be done for this anaemia? Natural erythropoietin is available but a patient would need 3000 units per day while total production in the United States in 1974 was only 50 000 units. It has not been synthesized. Large doses of oral or parenteral androgens may, on occasion, improve red cell production. She might have iron deficiency from red cell loss either during dialysis, from menorrhagia, or as a result of too many venepunctures. All patients on regular dialysis should have iron supplements. There is some impairment in iron absorption but patients can absorb enough from oral supplements. Folate deficiency may occur, but red cell folate levels are usually maintained and—though dialysis units usually give folate supplements—over eight years we have never given them and have never seen megaloblastic anaemia develop. Recently, histidine has been thought to be important by facilitating iron absorption, but there is little evidence that it improves anaemia. Finally, red cell survival is reduced in these patients. After nephrectomy this lady's anaemia was much worse, and red cell survival studies were carried out.

DR. A. J. W. HILSON (5): Red cell survival and splenic uptake studies carried out in July 1974 showed severe splenic sequestation and a red cell half life of 6·1 days. In a series of anephric patients on dialysis reported by Laurent *et al.* the half life was 18 days,[1] so that our patient's red cell half life was shorter than it should have been. Her splenic sequestration index was 175, which is considerably higher than the figure of 107 $\pm$ 19, which the same authors reported for anephric subjects.

DR. CATTELL: So she would have been expected to benefit from the splenectomy, as has been reported. Hypersplenism may be seen in 10% of patients on dialysis for over two years.

Post splenectomy she developed an "acute abdomen." Laparotomy showed a rupture in the right lobe of the liver. What is the explanation of this dramatic complication? She could have haemosiderosis, but I am not aware that this is a cause of rupture of the liver. Did she have serum hepatitis?

DR. OGG: I don't think so. She had negative tests for Australia antigen. In April 1974 she had a normal bilirubin and transaminase. But in retrospect her alkaline phosphatase which was then 30 K.A. units/100 ml had been rising for about a year.

DR. CATTELL: What about the hepatic angiography?

DR. H. M. SAXTON (6): There is a pack (shown by its markers) in the abdomen left to control the haemorrhage. All over the liver there are areas of abnormal circulation. Our interpretation was that there were multiple areas of tumour, though we had no idea of what type.

DR. CATTELL: I do not believe that this liver lesion is primarily a complication of her chronic renal disease or regular dialysis. But she was given a considerable amount of oestrogen, and I believe she has developed multiple adenomas of the liver, as has been reported after the prolonged use of hormone therapy.

Necropsy Findings

PROFESSOR LESSOF (7): I must congratulate you on a comprehensive analysis of the case. Would Dr. Brander give us the postmortem findings, with particular reference to the ruptured liver?

DR. W. L. BRANDER (8): The postmortem examination, which I attended, was carried out by Dr. Ian West on the instructions of the Southwark coroner. The liver weighed 4945 g (fig. 1).

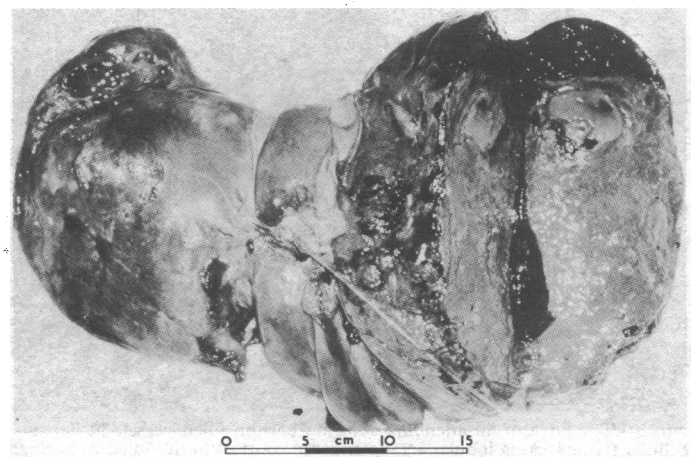

FIG. 1—The liver seen from behind with a vertical slice into the right lobe showing the subcapsular haematoma and several tumours on the cut surface. The cannula is tied into the left branch of the hepatic artery.

One vertical slice of the right lobe emphasized the size of the subcapsular haematoma, which extended over the superior and anterior surfaces of the right lobe of the liver. Postmortem arteriography via the left branch of the hepatic artery showed a central avascular area, outlined by leashes of vessels. There

were also other areas of abnormal circulation, which correlated well with the cut surface of the liver—which showed up to four tumours in the left lobe (fig. 2). The tumours were clearly demarcated from the surrounding liver but had no capsules. The cut surface was homogeneous compared with the lobular pattern of the adjacent liver. Histologically they were hepatic adenomata consisting of cords and plates of fairly normal looking hepatocytes separated by sinusoids, and containing numerous thin-walled, often barium filled arteries (fig. 3). So she had multiple hepatic adenomata, one of which had ruptured into the peritoneum. There was evidence of at least two episodes of bleeding from a similar tumour in the left lobe.

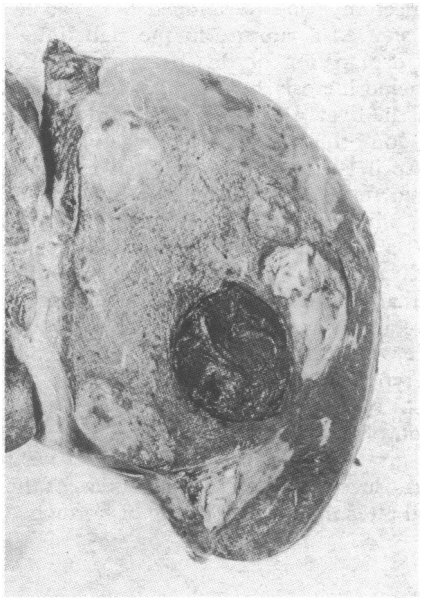

FIG. 2—Cut surface of the left lobe of the liver showing the central fresh haematoma displacing old haematoma laterally, and four adenomas.

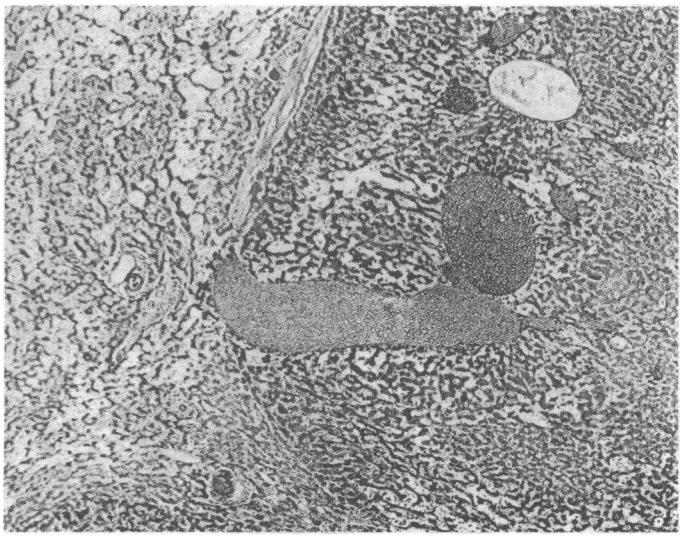

FIG. 3—The edge of an adenoma showing barium filled vessels in hepatocellular tissue lacking lobular arrangement in contrast to the adjacent normal liver. (Haematoxylin and eosin. × 25.)

Discussion

PROFESSOR LESSOF: Thank you. I would now like to call on Dr. Roger Williams to open the discussion. I would also like to congratulate Dr. Ogg for keeping his patient alive for 11 years with a disease which is not noted for its good prognosis.

DR. R. WILLIAMS (9): Yes, the only trouble is that she died of iatrogenic disease. In fact hepatic tumours in patients on the "pill" or who have had anabolic steroids in the past are not all that rare. Since 1973, when hepatic adenomata in women on the contraceptive pill were first described in the U.S.A.,[2] at least 18 cases have been reported and I know of at least four more in Britain. Usually the patients present, as did this one, with sudden intra-abdominal haemorrhage. The histology in the reported cases is that of a benign adenoma. Such bleeding into the peritoneal cavity is also seen with malignant hepatocellular tumours, particularly in the Far East, where hepatoma is common, and this is often the immediate cause of death. The benign tumours may bleed from vascular areas with histological appearances similar to those described for that very rare disease, peliosis hepatis, described in patients with tuberculosis.

One of our patients seen at King's College Hospital had areas of adenoma, other areas like peliosis, and one part that appeared malignant. She had had norethandrolone sometime ago and malignant tumours have been described in patients on this drug.[3] There is much discussion as to the link between malignant liver tumours seen in patients on androgens and the curious adenomas in women taking oestrogens, and our case at King's seems to bridge these two groups. Some adenomas have been successfully treated by resection but this patient under discussion was unfortunate to have had her whole liver involved. The duration of treatment with the pill has been very variable, sometimes as short as six months, though usually much longer. The condition may present after the pill has been stopped.

PROFESSOR LESSOF: The complications that a patient runs into when they survive under treatment for a serious disease raises a number of aspects. We have this patient's general practitioner, Dr. Baker, in the audience. Does he have any comments?

DR. J. W. BAKER (10): Thank you very much for inviting me. I had an extraordinary conversation with the patient's husband about three months after she died and I was so impressed by it that I dictated as much of it as I could remember.

The husband said, "I and my son are all right now and are off to Australia." I asked him about his wife and her death. He found it difficult to talk about it or describe it accurately. It was difficult for me because they had been an independent couple. As with most of the patients on home dialysis from the kidney unit at Guy's, they did not contact their G.P. very much but asked the unit directly if they had any trouble. This is not surprising in view of the complex technical nature of the machine they were managing. His comments were, "I came to hate that machine. My wife used to talk to it, you know, and so did I. I used to swear at it. It seemed to develop a personality of its own. It almost seemed to want to go wrong when we wanted to go out at night. So we rarely did because I had been up all night putting it right, or my wife had. She was very strong, you know. She had a lot of pain in her legs in the last year. Sometimes I found she had been up all night, but hadn't called me because I had to go to work the next day. She was very stubborn and much the stronger of the two of us."

What about the unit, I asked him. His comments were encouraging and they had both developed a strong relationship with the people in the unit. But he said, "She finally died because she was on the pill. They are very clever doctors up there, but I don't know that the pill was the right thing. I wouldn't go through it again, and I don't think my wife should have gone through it originally. It's a very difficult decision to make but that machine dominates you so much. I think unless a very firm assurance can be given to a patient that they are likely to be able to have a transplanted kidney in two years, they should think very seriously about whether an artificial kidney is worth it. It was a very miserable life for her, and for me, and for our son."

Now I don't think I agree with his conclusion. What I do feel, as a general practitioner, is that I failed in this family's terminal illness because I could not use my two main planks, early diagnosis and management of emotional reactions to disease. With patients on renal dialysis we do not have the repeated

continuous dealing with minor problems, which enables us to do this because it is taken over by the renal unit. They do it well, but I would like to make a plea for the greater involvement of the general practitioner with the renal unit and with long-term illness in our practices.

Is there a common ground, on a teaching and learning level, which renal units and general practitioners share, a ground which is not primarily an understanding of the disease process, but an understanding of how patients react to their disease process?

DR. OGG: I am delighted that Dr. Baker has said this. It took me two weekends to go through the notes, and when I saw what we had done to this poor girl I was horrified. The notes are a monument to her suffering. She had 34 admissions, 27 operations (including 18 for shunts), and 136 pints of blood (including 50 in the last few days). But I don't think we would make all these mistakes again. We have better access to the circulation, better and larger dialysers, better understanding of nutrition, less anaemia, and much less metastatic calcification.

We haven't yet solved the problem of menorrhagia, which crops up every day. But this woman did carry on as a housewife, and she had a little bit to spare because she also worked part time and contributed to local village life. And perhaps most important, her son had his mother's company for an extra seven years.

PROFESSOR LESSOF: Thank you. We have presented this case in a way that presents problems and sometimes our own inability to solve them.

This conference was recorded and edited by Dr. W. F. Whimster.

APPOINTMENTS OF SPEAKERS

(1) Dr. W. Cattell, M.D., F.R.C.P., Consultant Nephrologist, St. Bartholomew's Hospital, London.
(2) Dr. G. A. K. Missen, D.M., F.R.C.PATH., Consultant Morbid Anatomist, Guy's Hospital, London.
(3) Mr. T. M. Coltart, Ph.D., M.R.C.O.G., Senior Lecturer in Gynaecology, Guy's Hospital, London.
(4) Dr. C. S. Ogg, M.D., F.R.C.P., Renal Physician, Guy's Hospital, London.
(5) Dr. A. J. W. Hilson, M.B., M.R.C.P., Senior Registrar in Nuclear Medicine, Guy's Hospital, London.
(6) Dr. H. M. Saxton, F.R.C.P., F.F.R., Consultant Radiologist, Guy's Hospital, London.
(7) Professor M. H. Lessof, M.D., F.R.C.P., Professor of Medicine, Guy's Hospital Medical School, London.
(8) Dr. W. L. Brander, M.R.C.PATH., Lecturer in Histopathology, Guy's Hospital, London.
(9) Dr. R. Williams, M.D., F.R.C.P., Director, Liver Research Unit, King's College Hospital Medical School, London.
(10) Dr. J. W. Baker, M.R.C.G.P., D.OBST.R.C.O.G., General Practitioner, Addington Health Centre, Paddock Wood, Tonbridge, Kent.

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[3] Johnson, F. L., *et al.*, *Lancet*, 1972, **2**, 1273.

Medical Education

Postgraduate and Continuing Medical Education in a Developing Country

BRIAN SENEWIRATNE, M. KANAGARAJAH

British Medical Journal, 1975, **3**, 213-216

A major cause of frustration in young doctors in many developing countries is the lack of continuing medical education. This results in the emigration of many bright and enthusiastic students and is an important cause of the "brain drain."[1] Recently representatives from several developing countries claimed that the main reason for their doctors leaving was to seek higher education.[2]

Postgraduate education costs several times more than undergraduate education,[3] but can developing countries afford *not* to provide such an essential facility and almost certainly lose the best of their young doctors? If such countries, which are among the poorest in the world, are to get the best of the considerable investment they have made in training their own doctors some form of postgraduate instruction must be provided.

We outline the present system of postgraduate education (or lack of it) in Sri Lanka and suggest ways of improving it within the finances available. Any such programme must be implemented within the framework of the existing hospital system rather than by the establishment of a separate institute of postgraduate medicine, which would cost more than many developing countries can afford. Our observations are relevant to all developing countries.

University of Ceylon (Peradeniya Campus), Sri Lanka
BRIAN SENEWIRATNE, M.D., M.R.C.P., Senior Lecturer in Medicine
Kandy General Hospital, Sri Lanka
M. KANAGARAJAH, M.D., Physician

Present Hospital System

Sri Lanka has an area of some 25 000 square miles (64 750 sq km) and a population of 14 million. The hospital service is a tiered structure (fig. 1), with the Colombo group of 10 hospitals, which includes a teaching hospital, at the top. Then come the provincial hospitals (10), the one in Kandy being the second teaching hospital. Well-equipped libraries and well-qualified teaching staff in all the important specialties are available in the teaching hospitals. The provincial hospitals other than that in Kandy have similar specialized staff but no library facilities. Next come the base hospitals (12), where specialist staff are limited to a physician, surgeon, obstetrician, and, possibly, a paediatrician. Then come the district hospitals (96), which are run by a district medical officer (with no specialist qualification).